



Patient positioning for microdiscectomy nearly fatal because of iatrogenic obstruction to inferior cardiac inflow caused by hepatic hernia

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A routine microdiscectomy was complicated by an inferior cardiac inflow obstruction caused by compression of the inferior vena cava. This was due to further upward migration of an existing hepatic hernia through a right-sided diaphragmatic defect. Understanding the pathogenesis of this problem allowed the surgical team to adjust the positioning of the patient. After installing the patient on a different frame, the prone position was well tolerated and the microdiscectomy could be performed without the need to repair the diaphragmatic hernia.

Keywords : microdiscectomy ; diaphragmatic hernia ; compression inferior vena cava.

INTRODUCTION

The positioning of the patient for a surgical intervention is very important. If the patient is positioned and fixed correctly, the intervention can be made easier, reducing anaesthetic time and minimizing risks. Positioning the patient means manipulating a patient (with or without general anaesthesia) and therefore has inherent risks. The authors describe a case where this manipulation was almost fatal because of an undiagnosed right-sided diaphragmatic hernia with migration of the liver into the right hemithorax, causing obstruction of the venous return to the heart. They emphasize the importance of correct positioning with both surgeon and anaesthesiologist present in the operating theatre. Unexpected life threatening situations can

occur when previously missed disorders present themselves while the patient is under general anaesthesia or is positioned incorrectly. Immediate action may be necessary.

CASE REPORT

A 71-year-old obese woman (160 cm, 87 kg, BMI 33.98) was admitted with a right-sided herniated L4-L5 disc (fig 1) and a drop foot since 3 days. Her past medical history included allergy to penicillin, varicectomy, hysterectomy, cholecystectomy and type 2 diabetes mellitus. She did not recall any thoracic or abdominal trauma.

The following day, she was scheduled for a microdiscectomy under general anaesthesia. Anaesthesia induction was without any problem. Subsequently the patient was positioned prone on a "Wilson Frame" (fig 2) ; the surgical team, consisting of the orthopaedic surgeon, the orthopaedic registrar, the anaesthesiologist and the scrub nurse all helped. Within a minute after installation, the O₂ saturation

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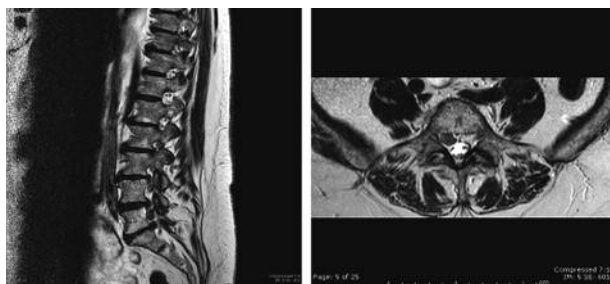


Fig. 1. — MRI-scan lumbar spine : a) T2-weighted sagittal view and b) T2-weighted axial view of the right-sided L4-L5 hernia.



Fig. 2. — Wilson frame

level dropped, not responding to good ventilation. Almost simultaneously the end tidal CO₂ dropped to 10 mm Hg (normally 35-40 mm Hg) and was followed by a drop in cardiac output. The patient was immediately turned back to the supine position, with rapid normalisation of the monitoring. The position of the endotracheal tube was checked.

A second attempt to position the patient was made with more monitoring (arterial line for the blood pressure), but the scenario repeated itself. The patient was taken to the recovery room and awakened. A thorough investigation was planned.

Sonography of the heart revealed mild insufficiency of the mitral and tricuspid valves, a small septum defect, but normal contractility of the left ventricle. A CT-scan of the abdomen and thorax showed a large mass in the right hemithorax (fig 3). An MRI-scan of the abdomen and thorax demonstrated that this mass in the right hemithorax was a part of the liver, herniated through a right-sided

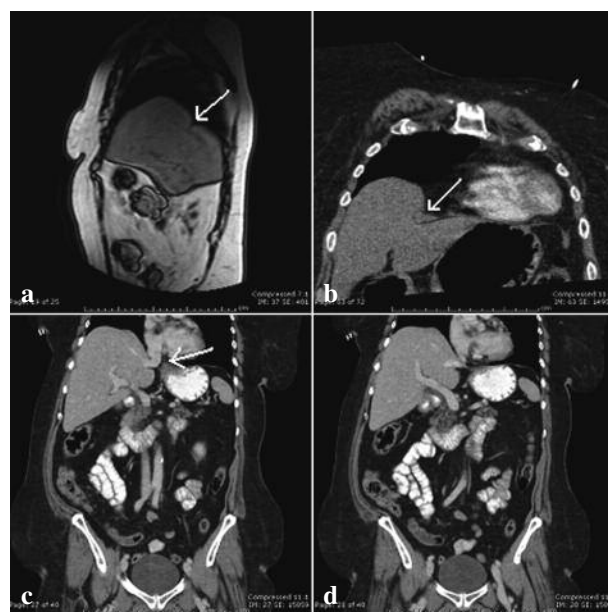


Fig. 3. — (a) MRI-scan of thoraco-abdominal viscera : sagittal view (white arrow shows rim of diaphragmatic defect with obvious indentation on the liver). (b) CT-scan viscera : coronal view of the herniated liver (white arrow indicates rim of diaphragmatic defect with obvious indentation on the liver). (c) CT-scan viscera : coronal view showing upwards migrating liver already bending the inferior vena cava (white arrow) with the patient lying supine. (d) CT-scan viscera : coronal view showing upwards migrating liver.

diaphragmatic defect. The borders of this defect were not frayed, suggesting that the hernia was not of a traumatic origin (fig 3). By positioning the patient prone on the “Wilson Frame” her abdomen had been compressed by the two side-bars of the frame, which had caused an increased abdominal pressure. This resulted in a further upward migration of the liver into the right hemithorax and compression of the inferior vena cava (IVC), so that the venous return to the heart through the IVC was subtotally blocked, causing an inferior cardiac inflow obstruction.

The patient, informed about the increased operative risk, consented to have the operation done on a different frame. This time she was placed prone on the fusion frame or “four-point” frame (fig 4). Her monitoring remained stable and a microdiscectomy could be performed without problems. Post-operatively her pain subsided rapidly and six weeks after surgery she regained ankle dorsiflexion.



Fig. 4. — Four-point frame mainly used for spinal fusions and scoliosis correction.

DISCUSSION

Diaphragm

The diaphragm is a complex structure, appearing between the fifth and the seventh week of gestation. Initially, the septum transversum forms a transverse partition that partially separates the coelomic cavity into an abdominal and a thoracic portion. There is however no absolute separation. The two cavities still communicate through two large dorsolateral openings : the pericardioperitoneal canals. Later on, the definitive musculotendinous diaphragm is formed. It incorporates derivatives of four embryonic structures : the septum transversum, the pleuroperitoneal membranes, paraxial mesoderm of the body wall and oesophageal mesenchyme. The septum transversum becomes the nonmuscular central tendon of the diaphragm. In congenital diaphragmatic herniations, one of the pericardioperitoneal canals fails to close and allows the abdominal viscera to bulge into the chest cavity. Depending on the mass of displaced viscera, the herniation blocks the growth of the lung on that side, which develops during the same embryological period. The left side is affected four to eight times more often than the right side, most probably because the left pericardioperitoneal canal is larger and closes later than the right. Congenital diaphragmatic hernia occurs in about 1 out of 2500 live births (9). There might even be an autosomal reces-

sive form of inheritance in total agenesis of the diaphragm (4).

Presentation of diaphragmatic hernia

Because of the complex development of the diaphragm, originating from four different embryonic structures, the clinical picture of a congenital diaphragmatic hernia has a wide spectrum on its own, and is sometimes combined with other congenital anomalies (4). The initial presentation highly depends on the location and dimension of the opening in the diaphragm (1). Due to improved prenatal ultrasound examination, more defects are now diagnosed before birth (4). Eighty to 90% of the congenital diaphragm defects identified at birth are complicated by hypoplasia or compression of the lung, giving life-threatening respiratory insufficiency. Four types of hypoplasia have been described, depending on the time the herniation occurs. This timing influences the clinical symptoms and the outcome : the earlier, the worse (16). The respiratory insufficiency caused by the hypoplasia can be aggravated by pulmonary hypertension, compression related atelectasis complicated with pneumonia due to hypoventilation, or decreased lymphatic drainage (13). If there is agenesis of the diaphragm, the patient does not always develop ventilatory insufficiency ; a possible reason could be that the hypoplasia of the lungs actually matches the ventilation and the perfusion (15). If so, the herniation is well tolerated and the diagnosis is often delayed. These 10 to 20% of the cases manifest later in life with a variety of respiratory (dyspnoea, tachypnoea, cough, recurrent respiratory infection, chest pain), gastro-intestinal (vomiting, abdominal pain, poor weight gain) or cardiovascular problems (1,12,13). These problems can be divided into acute, chronic and asymptomatic (3). Patients with right-sided herniations present themselves earlier than those with a left-sided herniation. There is a striking difference in the way left-sided and right-sided herniations present themselves clinically. These differences can be explained by the different viscera involved. If hollow viscera distend promptly or strangulate, there is an acute onset of symptoms. On the other hand, when the liver moves upward, which occurs

in most right-sided hernia patients, a clinical image of chronic complaints is present (1).

Differential diagnosis

The most frequent cause of hepatic hernia is a traumatic rupture of the diaphragm. Traffic accidents and falls from a height are mostly responsible for this kind of lesion. Both acute or late presentations do occur, with an incidence of 0.8 to 3.6% in blunt or penetrating thoracic, abdominal or combined thoracic-abdominal trauma. These lesions are often missed. Left-sided rupture is five times more frequent. In addition, half of the right-sided ruptures are treated late because of delayed diagnosis (7,8). If there is no history of trauma, the diagnosis of congenital diaphragmatic hernia or diaphragmatic agenesis is made by exclusion in asymptomatic patients. The first signs are often mistaken for some other pathology on the plain chest radiographs made preoperatively or during hospitalization. The left-sided hernia, with herniation of the stomach or the colon, is recognised easier than a right-sided hernia. A right-sided colon herniation can mimic several pulmonary processes, such as emphysematous bullae, pleural effusions and (retro-obstructive) abscesses (5,6). When the liver migrates into the thorax, it can mimic a tumour, which can be wrongly diagnosed as a peripheral cancer of the right lung (11).

Cardiovascular problems

Cardiovascular symptoms are very rare and can be caused by compression of the heart by herniated abdominal contents. In that case prompt surgical decompression and repair of the defect is needed. The pathogenesis is similar to that found in untreated tension pneumothorax, or, as in the authors' case, by blocking of the venous return to the heart when the liver is pushed cranially by increasing abdominal pressure, causing bending of the inferior vena cava (IVC) (2,12). Decrease in cardiac output and systemic effects have been described, with obvious difference between infrarenal and suprarenal obstruction of the IVC (10). Sudden cardiac arrest without preceding clinical symptoms has also been

reported, and the final diagnosis of congenital diaphragmatic hernia was made by autopsy in such cases (1). These cardio-vascular events without preceding clinical symptoms are often fatal. In the case reported here, the life threatening situation was corrected by turning the patient on her back, as described. In the supine position, the increased intra-abdominal pressure returned to normal, allowing the liver to migrate back to its original position in the abdomen. The IVC could straighten back out again, allowing the blood to flow back to the heart without obstruction.

Treatment

As most of the diaphragmatic herniae are already recognised before birth, with ultrasound scans during pregnancy, early surgical repair can be performed. Despite these advances in neonatal medicine however, congenital defects of the diaphragm still represent a major challenge with high mortality (1,4). Depending on the moment the defect is discovered, different types of treatment are used. Foetal interventions are still in an experimental phase. For the time being, temporary foetoscopic endoluminal tracheal obstruction is achieved by placing a temporary balloon in the trachea, allowing an accumulation of fluid in the lungs, which encourages better expansion of the lungs and decreases the effect of the lung hypoplasia. This balloon has to be removed before birth (13). To support the children who were born with lung hypoplasia, early intubation and treatment of pulmonary hypertension are started. ExtraCorporeal Membrane Oxygenation (ECMO) may also be used, as soon as the child is born. This ECMO allows the children to get an appropriate oxygenation despite the lung hypoplasia until the defect in the diaphragm is repaired. This ECMO consists of a machine connected to the patient by two lines, and oxygenates the blood of the patient as an artificial lung (4,13). In the children with late, but acute onset of symptoms, such as gastro-intestinal obstruction, prompt surgery is indicated through laparotomy, thoracotomy, thoraco-abdominal approach, laparoscopy or even thoracoscopy depending on the symptoms and the easiest way to gain access to the

acute problem correctly, while repairing the diaphragm through the same approach (1). This repair can be done with non-absorbable stitches. When the defect is too large to be sutured, or in case of agenesis, one can use a prosthetic mesh, a part of the fascia or a muscle flap from the abdominal wall (3,5). When the defect in the diaphragm is an incidental finding, and has not been symptomatic for years, treatment is debatable (1,14). A risk/benefit assessment should be made to compare the advantages of a repair with the possible risks of surgery.

In the authors' case the sciatica with drop foot was not life threatening. If the last attempt with the four-point frame had not worked, the patient would most likely have preferred walking with an orthosis, rather than taking the risk of major thoraco-abdominal surgery, followed by another attempt at microdissectomy, without any guarantee of neurological recovery.

Patient positioning

Once the diagnosis is made, if the prone position is necessary for an operation, it must be adapted to the special condition of the patient. Increasing the intra-abdominal pressure must be avoided at any price. A "Wilson Frame" is not ideal, as the side bars do not allow the abdomen to hang free. A "four-point" frame supports the thorax and the anterior superior iliac spines, leaving the abdomen free.

CONCLUSIONS

A right-sided diaphragmatic defect with herniation of the liver is a rare condition. This diagnosis can be missed easily when the patient has always been asymptomatic. Even when the preoperative chest radiographs reveal subtle signs, they can easily be mistaken for bullae, lung cancer or a higher right hemi-diaphragm. In the case presented, a herniation of the liver caused a life threatening situation when the patient was placed in the prone position on a Wilson frame. Thorough investigation allowed to make the diagnosis and to safely perform the microdissectomy in the prone position using a 4-point frame, thus avoiding pressure on the abdomen.

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